

three a day immediately postoperatively and she has had no blood in her stool since the operation. On May 23, 1950, sixteen months after operation, she returned for assessment. She had gained 30 lb., her stools while soft, did not occur more than twice a day and she has days when she has none. She looks and feels well. Sigmoidoscopic examination revealed ulcerative colitis.

In view of the result in this patient, one of our conservative internists, Dr. Frank Elliott, asked us to see another 26 year old girl with a similar type of history and similar findings. It is only a short time since she was operated upon but her immediate response was just as satisfactory. We realize full well these two cases mean little except that these two people have been greatly improved and have been returned so far to normal health.

Our very small series of cases of chronic ulcerative colitis is not of any value statistically or in the evaluation of surgery in this disease but McKell recently questioned 84 patients with permanent ileostomy. 79 were pleased with the results and said they wished it had been done earlier. McKittrick and Moore point out that 70 to 90% of their patients consider the end result of ileostomy satisfactory. These reports and our own small experience make one feel that surgical management in intractable ulcerative colitis is productive of better health than prolonged conservative treatment. I am convinced that many patients who now plan their daily activities from toilet to toilet could be made much happier with a properly functioning ileostomy and a satisfactorily fitting Koenig-Rutzen bag.

BIBLIOGRAPHY

1. CATTELL AND BOHE: *S. Clin. N.A.*, 26: No. 3, 1946.
2. THORLAKSON: *J. Internat. Coll. Surg.*, 12: No. 4, 1949.
3. COLCOCK, B. P.: *New England J. Med.*, 242: No. 9, 1950.
4. MILLER *et al.*: *Surg., Gynec. & Obst.*, 88: No. 3, 1949.
5. CAVE, H. W.: *Ann. Surg.*, 124: No. 4, 1946.
6. DENNIS, C.: *Surgery*, 18: No. 4, 1945.
7. DENNIS, E. *et al.*: *Ann. Surg.*, 129: No. 3, 1948.
8. DENNIS, C.: *South Dakota J. Med. & Pharm.*, March, 1949.
9. PORTIS: *J. A. M. A.*, 139: No. 4, 1949.
10. DIXON, AND BENSON: *S. Clin. North America*, 26: No. 4, 1946.
11. DRAGSTEDT *et al.*: *Ann. Surg.*, 114: 653, 1941.
12. ROWE, A. H.: *Ann. Int. Med.*, 17: 83, 1942.
13. FERGUSON, L. K. AND WELTY, R. F.: *S. Clin. North America*, 27: No. 6, 1947.
14. GRACE *et al.*: *J. A. M. A.*, 142: No. 14, 1950.
15. SLOAN, BARGEN AND BAGGENSTASS *et al.*: *Staff Meet. Mayo Clin.*, 25: No. 10, 1950.
16. BARGEN, J. A.: *The Management of Colitis*, Charles C. Thomas, Springfield, Illinois, 1944.
17. MCKITTRICK AND MOORE: *J. A. M. A.*, 139: No. 4, 1949.
18. KIEFER, E. D.: *J. Omaha Midwest Clin. Soc.*, January, 1942.
19. MCKELL: Quoted by Kiefer.

INTERNAL MAMMARY CORONARY ANASTOMOSIS IN THE SURGICAL TREATMENT OF CORONARY ARTERY INSUFFICIENCY*

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THIS paper constitutes a preliminary report of clinical cases which have undergone transplantation of the left internal mammary artery into the left ventricle as a treatment for coronary artery insufficiency. The theoretical and experimental basis on which this procedure is based will be briefly described.

EXPERIMENTAL DATA

Many attempts have been made experimentally to improve ventricular myocardial circulation such as, the application of fat or muscle grafts to the heart, the use of irritating foreign bodies such as talc, or asbestos fibres to produce adhesions between pericardium and myocardium. Fauteux¹ attempted to improve the myocardial circulation by cardiac vein ligation and Beck² by means of arterialization of the coronary sinus. One of us (A.V.) has attempted to obtain this improvement by the direct implantation of a living artery, namely the left internal mammary artery into the left ventricular myocardium. The artery is placed within a tunnel in the myocardium and in over 200 experiments the degree and frequency of new anastomoses have been experimentally proved by injection studies, radio-graphs, plastic casts and serial sections.^{3, 4} Anastomosis occurred in 50 to 75% of these animals, depending on the technique of implantation used. No animal developed infarction or died following anterior descending branch ligation when a large anastomosis had developed. All these experiments have been controlled and the control group, without implantation of a living vessel into the heart muscle, showed a mortality of 90%, and in 10% a large infarction developed following the same ligation of the anterior descending branch.^{5, 6} (It is recognized that thrombosis of the anterior descending branch of the left coronary artery is the most common cause of death in human coronary artery disease.)

The anastomoses which developed have been shown both by injection and by histological serial section to be true arterial branches. It has been reported by Glenn⁷ that these branches only live for 8 weeks. Our observations have definitely shown that they last at least 58 weeks which is the longest observation made before the animal was sacrificed to confirm the persistence of these vessels.⁸ The direction of blood flow through the implanted internal mammary artery was studied in order to determine whether or not blood was being brought to the ventricular myocardium through the internal mammary artery. Direct determination of blood flow in the internal mammary artery was difficult, so the indirect method was used. Animals which survived anterior descending branch ligation of the left coronary artery were subjected after 4 weeks to complete and sudden occlusion of the implanted internal mammary artery. If the internal mammary artery was maintaining the circulation of the left ventricle then, following its ligation, either death or infarction should result. This is exactly what happened. In three animals with an internal mammary implant that had survived anterior descending branch ligation, the internal mammary artery was ligated. One animal died within 24

The more things a man is ashamed of, the more respectable he is.—George Bernard Shaw.

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hours and displayed an oedematous cyanotic area of the anterior wall of the left ventricle. One survived for 3 days before dying from a large infarct in the same location. The 3rd animal survived, but examination of the sacrificed specimen revealed that multiple intercoronary anastomoses were present.

The experimental results just described were obtained by implanting the internal mammary artery into normal dogs' hearts. It was suggested that in human coronary insufficiency such a procedure would be of little value because of the presence of occluded coronary vessels and associated myocardial ischaemia. It was, therefore, decided to experimentally produce coronary artery insufficiency in the dog. This was done by wrapping the origin of the anterior descending branch of the left coronary artery with a sclerosing type of cellophane.⁹ The cellophane surrounding the origin of the anterior descending branch of the left coronary artery set up a periarterial fibroplasia which resulted in a gradual contraction through scar tissue of the coronary artery with narrowing of its lumen. This caused a reduction in blood flow through the narrowed vessel, and resulted in ischaemia of the left ventricular muscle supplied by the anterior descending branch of the left coronary artery. The degree of myocardial ischaemia was evaluated by estimating the exercise tolerance of the animal on a motorized treadmill. Before the cellophane wrap was placed around the coronary artery the animals would run 9 to 12 minutes at 8½ miles per hour on the treadmill. With this amount of exercise the animals became tired, began to lag on the mill and would break alternatively from a gallop to a run. Eventually they would become anxious to escape the mill. When the exercise was discontinued these animals would pant, but would drink water and appear similar to any other normal dog after exercise. Five months after these animals had had a piece of cellophane wrapped around their anterior descending branch of the left coronary artery they would run for 1 6/10 minutes on the treadmill before becoming extremely anxious, begin to whine and salivate profusely. If the exercise was not terminated they would attempt to lie down or drag their feet on the revolving platform. When the mill was stopped they would drop where they stood and resist all coaxing to move for some minutes and would not drink water. Animals that had reached this stage were then subjected to a left internal mammary artery implant. Four months after the implant those animals which had definitely developed an internal mammary coronary anastomosis had a return of exercise tolerance to 7 minutes or more. This occurred in spite of a completely occluded anterior descending branch of the left coronary artery. When an internal mammary coronary anastomosis failed to develop there was no improvement in exercise tolerance in such animals after implantation.

Because of these experimental results it was thought that implantation of the internal mammary artery into the left ventricular myocardium might be of value in the treatment of human cases of coronary artery insufficiency.

HUMAN CASES OF CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY TRANSPLANT

Selection of cases.—The estimation of clinical results is always difficult and the results of any given surgical procedure may vary according to the severity of the disease process at the time of operation. There are certain well-known pathological facts concerning coronary artery sclerosis and thrombosis which greatly influence the selection of cases for internal mammary artery implant. Perhaps the most important is the fact that coronary artery sclerosis in gen-

eral is confined to the first 3 or 4 cm. of the coronary artery. It has been stated that arteries beyond the first 3 or 4 cm. of the coronary vessels show lesser degrees of sclerosis and rarely is sclerosis seen after the 3rd or 4th order of branching. Sections have shown that in cases of severe coronary artery sclerosis the vessels lying within the heart muscle are generally free of arterial disease. Thus, an internal mammary artery placed in the ventricular muscle is placed in an area where the arteries are comparatively healthy. In this way fresh blood can be brought to the network of non-sclerosed arteries and arteries which exist beyond the points of coronary artery obstructions.

In cases of coronary artery thrombosis with myocardial infarct,¹⁰ the picture is entirely different. In these cases, if the patient survives, an area of myocardial infarction the muscle undergoes degeneration and eventually heals by scar formation. In most cases there will be healthy muscle surrounding the area of the healed infarct. It is also stated that during the process of healing new blood vessels grow into the infarcted area; thus the history of a left coronary artery thrombosis with recovery does not constitute a contraindication to internal mammary artery implantation. The new living arteries can be placed in healthy muscle which is present at the edge of the healed infarct and, if necessary, into the intraventricular septum itself. With this in mind, it is clear that patients who have recovered from coronary artery thrombosis and infarction may be considered as candidates for an internal mammary artery transplant. Our last patient was known to have had two attacks of coronary artery thrombosis with a posterior wall infarct. At operation there was still what appeared to be good muscle posteriorly. There was, however, evidence of scarring on the posterior surface towards the apex which extended for a half inch or more to the anterior surface of the left ventricle. The internal mammary artery was placed into healthy muscle which was present just proximal to this scarred area. We have been reluctant to operate upon patients who are able to carry on their normal daily activities. We have to date only operated upon those patients who are unable to carry on because of the severity of their anginal pain. Patients with an enlarged left ventricle and myocardial decompensation have been considered poor risks and have not been accepted. Every attempt has been made to exclude other sources

of anginal pain and to make certain that the pain from which the patient is suffering is due to coronary insufficiency. Where there is a doubt and particularly when other organic disease such as cholelithiasis exists, the associated disease has been treated first.*

Preoperative investigation.—Each patient has undergone extensive investigation in order to establish a diagnosis of coronary insufficiency and to determine its extent. A careful clinical history has been supplemented by detailed electrocardiographic studies made at rest and after exercise. In order to correlate the anginal pain with myocardial ischaemia electrocardiograms have been taken after exercise at the height of the anginal pain. A record was made on each patient as to the extent of his exercise tolerance prior to operation as indicated by the number of stairs he could climb in a given time before experiencing the onset of anginal pain.

In each patient a careful survey has been made to exclude sources of pain which were not cardiac in origin. Complete radiographic studies have been made of the oesophagus, stomach, duodenum and gallbladder, as well as of the thorax and lungs. In each case a Mosenthal test, non-protein nitrogen, basal metabolic rate, blood cholesterol, blood sugar and haemograms were also recorded.

CASE 1

Mr. J.P., (referred by Dr. L. I. Frohlich of Montreal) age 53, occupation, tailor. Admitted to the Royal Victoria Hospital, April 24, 1950. On admission the patient's chief complaint was that of substernal pain which radiated to the left jaw and down the left arm to the wrist. Occasionally the pain radiated to the back in the interscapular region. The pain was brought on by exertion and emotion. It was an aching, pressure type of pain, and was not very sharp. It was first noticed 14 years ago, gradually becoming more severe. The patient had been unable to work for three years prior to admission and had suffered pain day and night. Some, but not complete, relief was obtained by nitroglycerin. Exercise tolerance was limited to one city block; walked very slowly before onset of anginal pain occurred. There was a history of 40 pounds loss of weight in the past 10 years. There was also a history of intolerance to fatty foods with eructations of gas and inability to eat a large meal.

General physical examination revealed a well nourished white male of good colour. Temperature was 98, pulse 80, respiration 20, blood pressure 120/90. There was a right, direct inguinal hernia. There were bilateral varicose veins.

Exercise tolerance test.—The patient developed pain after going up 22 steps in a 10 minute period. The electro-cardiograms, taken before and after exercise, are shown in Figs. 1 and 2 respectively. Blood and spinal fluid serology were negative. The lungs, oesophagus, stomach, duodenum and gallbladder were radio-

logically normal. Blood analysis for sugar and cholesterol were found to be within normal limits. There was no increase in white blood count or the sedimentation rate.

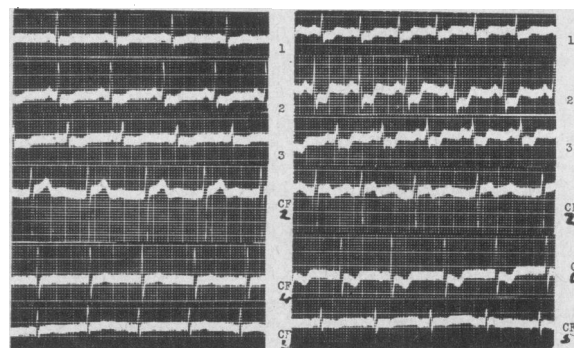


Fig. 1

Fig. 2

Fig. 1.—Electrocardiogram taken after 3 hours' rest in bed. A slurring of QRS complexes. Depression of S-T interval in leads 1, 2 and 3. Lead CF₁ normal. CF₂ shows a low T-wave as does CF₃. Myocardial changes with coronary insufficiency. Rate 60 per minute. **Fig. 2.**—Electrocardiogram taken at height of pain after 22 steps climbed in 10 minutes. The standard leads show a marked depression of the S-T intervals after exercise. A marked coronary insufficiency. The CF leads show a diphasic T-wave. CF₁ a negative T and a shallow negative T in CF₂. Evidence of marked coronary insufficiency. Rate 90.

Operation.—On April 28, 1950, implantation of the left internal mammary artery into the left ventricular myocardium was carried out. Prior to the commencement of the anaesthetic, the patient experienced severe substernal pain which was not relieved by two tablets of nitroglycerin. An electrocardiogram taken at this time did not show any changes indicative of coronary thrombosis. The blood pressure was unaltered so it was decided to proceed with the operation. The left thorax was entered through the 5th intercostal space by an anterolateral approach. The 4th and 5th ribs were resected subperiostally 10 cm. lateral to the sternum, including part of their cartilages. Approximately 1.5 cm. of cartilage was left in position. Procaine 1% was injected into the pericardium and was also given intravenously as a continuous drip. At this time the blood pressure, which had slowly been dropping, was recorded at 70/50. Patient was placed in the Trendelenburg position and the blood pressure returned to 90/70. The internal mammary artery was freed from the chest wall between the 4th and 6th intercostal spaces. The intercostal arteries 4th and 5th were doubly ligated with 000 catgut. The internal mammary artery was tied with cotton and severed between ligatures. The pericardium was opened. The left ventricular muscle was firm and was covered by a layer of fat. A traction

* The final decision in the selection of each patient for operation was made jointly with Dr. G. R. Brow, Director of the Department of Medicine.

suture was placed in the apex of the heart. The 6th intercostal artery was cut and bled freely and was pulled with the internal mammary artery into a tunnel in the myocardium. The internal mammary artery was held in position by a traction suture which was tied about it. The entire procedure of implantation took about 3 minutes. There was little evidence of ventricular irritability or arrhythmia. The blood pressure at the time of implantation was 70/60 which quickly returned to 110/80 after the thorax was closed. The pulse rate throughout the operation was comparatively slow at about 100 per minute. The left thorax was drained. The total blood loss during operation was 260 c.c., as measured by the gravimetric method.

Postoperative course.—For the first 12 hours after operation the blood pressure remained steady at 110/72 with a pulse rate of 108 per minute. The patient was conscious and appeared clinically quite well. Gradually the blood pressure sank until 24 hours after operation it was 86/62. The pulse rate, however, had dropped to 96 per minute and the patient's general condition was excellent. He was removed from the oxygen tent and given a liquid diet. An electrocardiogram taken approximately 24 hours after operation showed anterior myocardial changes and coronary insufficiency, but the rhythm was regular, and the a-v conduction time was normal. In spite of the low blood pressure the patient's general condition remained excellent throughout the succeeding two days. At 5.54 p.m., April 30 he attempted to use the bed pan. This was followed by a rapid drop in blood pressure and elevation of pulse rate to 160 per minute. On May 1, at 12.30 a.m., chest pain developed and there was an increase of the respiration rate to 22. At 1.25 a.m. the chest pain became more severe and the condition of the patient rapidly deteriorated. He expired at 1.45 p.m.

The interval between completion of operation and death was a little over 2½ days.

PATHOLOGICAL FINDINGS

The three main coronary arteries were pipe-stem in character for their first 3 or 4 cm. All the coronary arteries showed multiple areas of marked stenosis due to arteriosclerotic plaquing. The right coronary artery was completely blocked by an old, firm, greyish thrombosis. The left anterior descending branch was completely occluded for a distance of 1 cm. by a recent, soft,

dark-brown thrombosis. The anterior wall of the left ventricle and intraventricular septum showed evidence of recent infarction.

The internal mammary artery which had been implanted was patent throughout. There was no hæmatoma at the site of implant (see Fig. 3).

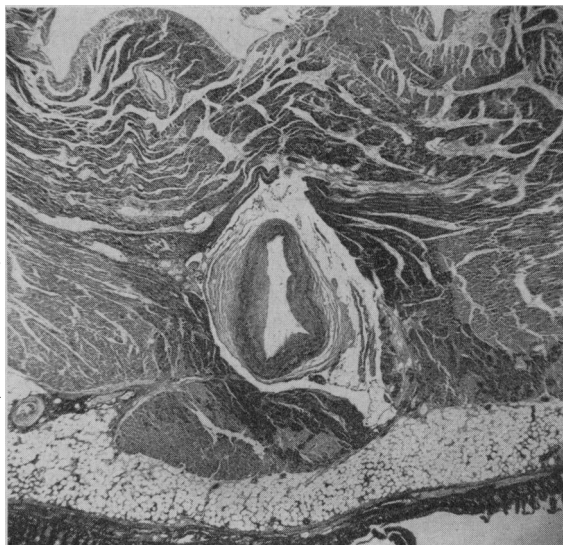


Fig. 3.—Shows the internal mammary artery lying in the human myocardium 62 hours after implantation.

Death was due to a recent thrombosis of the anterior descending branch of the left coronary artery with ventricular infarction. There was an associated atelectasis of the left lower lobe.

CASE 2

Mr. D.M., age 54. Admitted to the Royal Victoria Hospital, October 22, 1950. Discharged December 5, 1950. This patient, 7 months prior to admission, developed severe precordial pains which radiated down the left arm. Pain was initiated by exercise and was associated with shortness of breath. Pain frequently followed the ingestion of solid food. This was so pronounced that for a few months prior to admission patient lived on a liquid diet. His exercise tolerance was limited to about one city block. There was a bad family history. His father and two paternal uncles died of heart disease. One brother had had coronary artery thrombosis.

Physical examination revealed a well nourished slightly obese adult male with sallow complexion and a somewhat myxœdematous appearance to his face. The abdomen was pendulous due to excessive fat. Pulse 68 to 74, temperature 98, respiration 18, blood pressure ranged between 140/100 to 106/60.

Exercise tolerance.—Fifty-six steps with 7" elevation were climbed in 2½ minutes before pain in the precordium and left arm occurred. A control electrocardiogram taken at rest is shown in Fig. 4, and an electrocardiogram exercise taken at the height of pain is shown in Fig. 5.

Blood serology and chemistry for sugar and non-protein nitrogen were normal. Radiographic studies of the lungs, œsophagus, stomach, duodenum and gall-bladder were normal. There was no elevation of the white count or sedimentation rate. The Mosenthal test showed excellent concentration and output. The basal metabolic rate was normal.

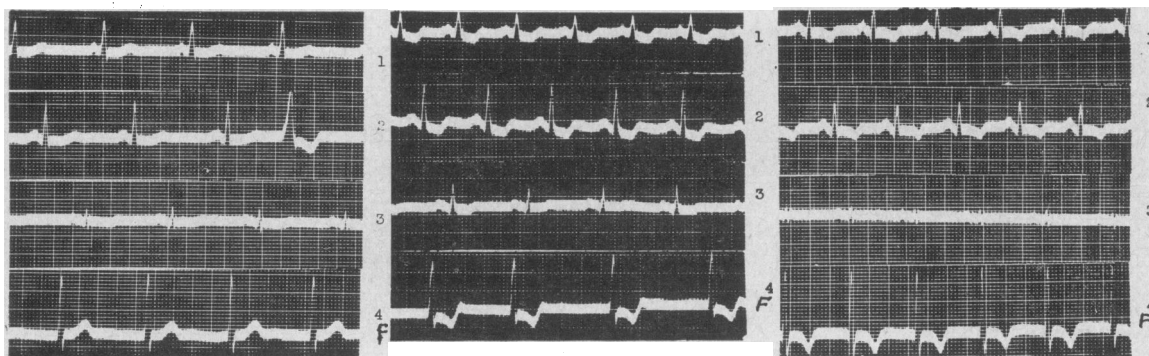


Fig. 4

Fig. 5

Fig. 6

Fig. 4.—Sinus rhythm. Normal auriculo-ventricular conduction time. QRS complexes slurred. Right ventricular extra systoles in lead II. Low voltage T-waves in all leads. Diphasic P-waves in lead III. Ventricular rate 60 per minute. Lead 4F adds nothing more. **Fig. 5.**—Regular rhythm. Normal auriculo-ventricular conduction time. QRS complexes slurred. R-T intervals depressed in leads I and II. Diphasic T-waves in lead III. Ventricular rate 90 per minute. Lead 4F shows a depressed S-T and negative T-waves. Coronary insufficiency. **Fig. 6.**—Regular rhythm. Normal auriculo-ventricular conduction time. Negative T-waves in leads I and II. QRS complexes slurred. Ventricular rate 90 per minute. Lead 4F shows an acutely inverted T-wave. The picture is that of recent anterior infarction.

Operation.—In this patient and in the subsequent case, the blood pressure was maintained throughout the operation by means of a continuous intravenous drip of neosynephrin. The blood pressure was 130/60 at the commencement of the anæsthetic and was never permitted to drop below 120/70 throughout the entire operation which lasted approximately 2½ hours. It was necessary to increase the rate of flow of neosynephrin at various times during the operation in order to prevent the blood pressure from dropping. The technique followed was approximately the same as that described in the first case, except for the approach which was made through the 5th intercostal space with severance of the 4th and 5th ribs just distal to the costal cartilages. The heart at the time of implant showed little disturbance. The blood pressure was maintained throughout. There was no fall in blood pressure during the operation such as occurred in the first patient. The anæsthetic used was cyclopropane and ether.

Postoperative convalescence.—The immediate postoperative blood pressure was well maintained and varied from 142/106 to 100/70. At the time of discharge, the blood pressure varied from 140/80 to 100/74. The pulse rate 48 hours after operation reached 120 per minute. This slowly diminished, and at time of discharge was between 80 and 90. The postoperative convalescence was essentially uneventful except for the complication of a paralytic ileus which was easily controlled by Wangenstein drainage.

There was also a diffuse pleuritis of the left thorax which gradually improved.

The patient was allowed to sit on the side of the bed on the 10th postoperative day, and was permitted to sit in a chair on the 23rd day. He was discharged to his home five weeks after operation. At the time of his discharge he was able to eat solid food and walk slowly. There was no anginal pain after eating or during walking. An electrocardiogram taken the day before discharge is shown in Fig. 6. Recent communication from this patient states that he is completely free of pain and working.

CASE 3

Mr. E.S., age 49. Admitted to the Royal Victoria Hospital, November 9, 1950. Discharged December 18, 1950. This patient, in December, 1947, developed uræmia and was extremely ill. In 1948 after playing 27 holes of golf, he experienced severe pain in his left arm which radiated to the chest. A diagnosis of left posterior branch coronary occlusion was made. Since that time he has suffered from anginal pain which radiated up his left arm to the precordial region and sternum. Occasionally, the pain went into the throat and jaw. The anginal-like pain was initiated by any change of pace. It was particularly severe on getting out of bed in the morning or on walking rapidly. It was relieved by nitroglycerin. Sometimes it was so severe that patient was forced to take demerol.

Temperature was 97.2, pulse rate varied from 112 to 90, respirations 18, blood pressure varied from 174/120 to 120/90. Physical examination was essentially normal.

Exercise tolerance.—Ninety steps were taken in 1½ minutes without increasing the pain. The pain, however, was present before starting the exercise. Control and exercise electrocardiograms are shown in Figs. 7 and 8. Blood chemistry for serum cholesterol, sugar and non-protein nitrogen were within normal limits. The basal metabolic rate, hæmogram and Mosenthal test were normal. Radiographic examination of the lungs, œsophagus, stomach, duodenum and gallbladder were normal.

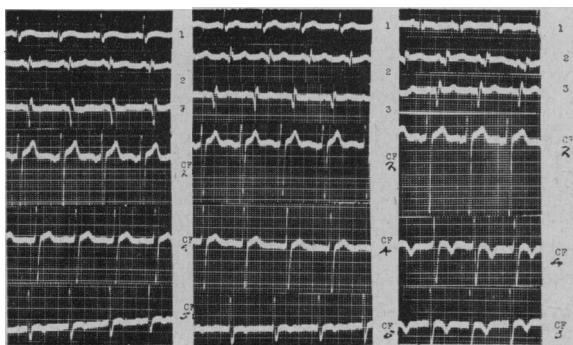


Fig. 7

Fig. 8

Fig. 9

Fig. 7.—Regular rhythm. Normal auriculo-ventricular conduction time. Left-sided preponderance. QRS complexes slurred. Q-wave in leads II and III. T-wave flattened in all standard leads. Ventricular rate 90 per minute. Leads CF₁ and 4 add nothing. CF₁ shows negative T-waves. Myocardial changes. Coronary insufficiency. **Fig. 8.**—Regular rhythm. Normal auriculo-ventricular conduction time. QRS complexes slurred; Q-waves in leads II and III. T-waves of greater voltage. Ventricular rate 90 per minute. Leads CF₁, CF₂ and CF₃ are unchanged. **Fig. 9.**—Regular rhythm. Normal A-V conduction time. Left-sided preponderance. QRS complexes slurred. Q waves in leads 2, 3. Negative T-waves with elevated S-T in lead 1. R-T elevated in lead 3. Ventricular rate 90/min. Leads CF₁ and CF₂ show an acutely negative T-wave. Anterior myocardial infarction.

Operation.—On November 20, 1950, under cyclopropane and ether, an internal mammary artery implant was carried out. The blood pressure at the commencement of the anæsthetic was 130/80, and was quite difficult to maintain before and during operation. Large amounts of neosynephrin were given in order to keep the blood pressure at approximately 130/80. A total of 4 c.c. of 1% neosynephrin was used in this case from the time the anæsthetic was commenced to the conclusion of the anæsthetic. The pulse rate at the beginning of the operation was 110 per minute. It climbed to 140 and 150 per minute and then settled down to approximately 130 per minute for the duration of the procedure. The operative technique followed was essentially the same as in the second case, except for the fact that the 5th rib and costal cartilage were removed, which facilitated exposure. The implantation of the left internal mammary artery into the myocardium of the left ventricle caused little cardiac disturbance. It should be noted here that in this case and in the second case the 6th intercostal arteries were not bleeding at the time of implant.

Postoperative convalescence.—The pulse rate reached 130 at the end of 48 hours. This slowly settled down to vary between 80 and 100 where

it was at the time of his discharge. The immediate postoperative blood pressure varied between 142/100 to 100/72. This patient developed a paralytic ileus and a patch of pneumonia in the right lower lobe. Paralytic ileus was treated by means of gastric and duodenal decompression and the pneumonia with streptomycin and aureomycin. The patient was given 3 grains of quinidine before and after operation. He was permitted out of bed at the end of three weeks and returned to his home four weeks after the operation. At the time of his discharge he showed marked improvement of his anginal pectoris. There remained only a slight pain in the left wrist upon getting out of bed in the morning. This did not require nitroglycerin or demerol. The discharge electrocardiogram is shown in Fig. 9.

DISCUSSION

In our experimental work it has been shown that when the internal mammary artery is placed in the ventricular myocardium it forms new arterial branches which anastomose with the left coronary circulation. When this occurs, the heart is protected against death by infarction following the occlusion of the anterior descending branch of the left coronary artery. It has also been shown that those animals which have survived a ligation of the anterior descending branch of the left coronary artery die or develop infarction when the implanted internal mammary artery is occluded. An internal mammary coronary artery anastomosis has been shown to be of functional value. It has relieved artificially produced coronary artery insufficiency. It is reasonable to assume on the basis of our experimental work and the pathological facts of coronary artery sclerosis that an internal mammary artery implant may be of value in the treatment of human coronary artery insufficiency.

Cases have to be carefully selected and treated by a medical-surgical team, and it is our opinion that the use of quinidine pre- and post-operatively with procaine during the operation is important in preventing ventricular fibrillation. Our first patient, we believe, developed his coronary thrombosis because of the continuous low blood pressure which was present throughout the operation. In order to bring these patients through an intra-thoracic operation without further damaging their coronary artery system,

we believe it is necessary to maintain their blood pressures above 100 mm. Hg. throughout the entire operative procedure.

The postoperative care of these cases is fundamentally a medical problem. Unlike other thoracic cases, these patients should not be moved frequently in the first few postoperative days. It would seem best to treat them postoperatively much in the same manner as a case of coronary artery thrombosis. Interpretation of postoperative electrocardiographs is difficult because of the disturbances created by the implantation of a pulsating artery into the anterior wall of the left myocardium. Clinically there has been no evidence of coronary artery thrombosis developing after operation in the two cases which have survived. It is too early to estimate results.

SUMMARY

1. The internal mammary artery can be implanted in the ventricular myocardium in man with recovery.
2. There appears to be no disturbance in cardiac function resulting from the implant procedure.
3. The internal mammary artery in man, as in the animal, was found to be completely patent 62 hours after implantation in the one fatality that occurred.
4. In spite of the burying of an open vessel in the myocardium, there was no evidence of hæmorrhage or intramural hæmatoma.
5. The last two cases appear to have been improved at the time of discharge.

We wish to express our appreciation for the counsel, criticism and continued support which have been given by Drs. Lyman Duff, Donald Webster and C. A. MacIntosh. In particular do we wish to thank Dr. G. R. Brow for his careful selection of cases for operation and for his help in their postoperative care.

REFERENCES

1. FAUTEUX, M.: *Surg., Gynec. & Obst.*, 71: 151, 1940.
2. BECK, C. S. AND TICHY, V. L.: *Am. Heart J.*, 10: 849, 1935.
3. VINEBERG, A. M.: *Canad. M. A. J.*, 55: 117, 1946.
4. VINEBERG, A. M. AND JEWETT, B. L.: *Canad. M. A. J.*, 56: 609, 1947.
5. VINEBERG, A. M.: *J. Thorac. Surg.*, 6: 839, 1949.
6. VINEBERG, A. M. AND NILOFF, P. H.: *Surg., Gynec. & Obst.*, 91: 551, 1950.
7. GLENN, F. AND BEAL, J. M.: *Surgery*, 27: 841, 1950.
8. VINEBERG, A. M., NILOFF, P. H. AND MILLER, D.: *Proc. Royal Coll. Physicians and Surgeons of Canada*, Montreal, December, 1950. In Press.
9. MILLER, D. AND VINEBERG, A. M.: *Proc. of Surgical Forum, American College of Surgeons, Boston*, October, 1950. In Press.

BOOKS ON ART FOR WAR-DAMAGED LIBRARIES.—In response to a Unesco appeal for art books and prints to war-damaged libraries, the San Francisco Museum of Art has sent material of this kind to Austria, Czechoslovakia, Germany, the Netherlands and Poland. Subscriptions to various American art periodicals have also been donated.—(UNESCO.)

AMNIOTIC FLUID EMBOLISM*

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SINCE the first 8 cases of amniotic fluid embolism were described by Steiner and Lushbaugh¹ in 1941, 14 additional cases have been reported, thus justifying the authors' prophecy that this condition would be found to be an important cause of maternal mortality. They estimated its incidence to be once in every 8,000 confinements, and said it was responsible for 0.2% of all maternal obstetrical deaths. In a recent report in 1949, the same authors¹³ altered their estimate of its incidence to once in every 20,000 confinements.

Because we understand so little of the mechanism of death in amniotic fluid embolism and because of the lack of prodromal symptoms, little has been accomplished in its prevention. In the present paper an additional case is reported, previous cases are reviewed, and the significance of the finding of blood incoagulability is discussed.

CASE REPORT

A 39 year old woman had had three normal, full-term deliveries in 1935, 1938 and 1946, respectively, and these infants had varied from 8 lb. 14 oz. to 7 lb. 14 oz. in weight. Three other pregnancies in 1943, in 1944 and in 1949 had terminated in abortion for unknown reasons. During hospitalization following the last abortion in 1949, she required transfusion for blood loss.

During her seventh pregnancy, the patient was carefully followed in prenatal clinic. The date of expected delivery was April 8. Physical examination revealed no abnormality. The blood Wassermann reaction was negative, and the blood group was O, Rh positive. Urinalyses revealed a faint trace of albumin on five occasions, and a faint trace of sugar on one occasion. At the last prenatal visit, 13 days before admission to hospital, the baby was active, but the fetal heart was inaudible.

The patient was admitted to hospital on April 15 (at 41 weeks) in the second stage of labour. Spontaneous rupture of the membranes had occurred half an hour previously. Labour pains came on every two to three minutes, and were of such severity that the patient had difficulty in walking from the automobile into the hospital. The cervix was well dilated, the fetal head well down in the pelvis, the presentation vertex, right occipito-posterior, and the fetal heart inaudible.

One and a half hours after admission, the patient was anesthetized with ether and oxygen, following induction by nitrous oxide and cyclopropane. Manual rotation was performed, and a dead, 9 lb. 12½ oz. baby was delivered by low forceps, after removal of a loop of cord from around the infant's neck. The patient remained in excellent condition during this procedure.

Twenty minutes after delivery, during repair of the episiotomy, before delivery of the placenta, the patient suddenly became profoundly shocked, her blood pressure

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